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ANÆSTHESIA FOR THE TUBERCULOUS PATIENT*

BY

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JUST as the Younger Pliny said: "I am one of those who admire the ancients, yet I do not, like some others, under-rate certain instances of genius which our own times afford. For it is not true that nature, as if weary and effete, no longer produces what is admirable." So prudent thought chooses the middle course between Romanticism, or urge, and Classicism, or restraint. Thus, in anæsthesia, of importance are those precepts which belong to the selection of the anæsthetic agents and the methods of their administration.

It is when the physician, the surgeon and the anæsthetist discuss together the general physiological state of the patient, as well as the surgical requirements of the case, that the best choice is made. With earnest of mind, these three medical philosophers will choose the drugs to be used and the manner of their employment, after considering *the nature of the operation that must be done*, and *the character of any additional disability*, while being fully aware of the harmful effects which such drugs are capable of producing. From

* Read at a meeting of the Section on Tuberculosis of the Montreal Medico-Chirurgical Society, January 21, 1942.

this general injunction there may be no exception, even for the tuberculous patient.

Henry David Thoreau has written in his *Walden* that—"In the long run men hit only what they aim at. Therefore, though they should fail immediately, they had better aim at something high." In like manner, we should endeavour to produce the most efficacious anæsthesia with the least harmful effects and with the greatest degree of comfort, even to the healthiest person. I see no good purpose in abusing the robust—no good reason why the strong man who does our fighting should not have the best when he must be given an anæsthetic. But, as it is customary to make special considerations for the handicapped patient, we must constantly keep before us certain salient features in the selection and administration of anæsthetics for such individuals as suffer from tuberculosis and need to be operated upon. No matter what the procedure, it is particularly essential that a free and unobstructed respiratory air-way be maintained throughout, as the tuberculous patient ought to be allowed an abundance of oxygen. In choosing the course of action, the following principles should be observed. (1) The anæsthetics used should not irritate or damage the lung. (2) The reflexes should be depressed as little as possible, in order that coughing may not be hindered too long. (3) The drugs used should be those which cause the least disturbance to metabolism. (4) The method should permit a sufficiency of oxygen. These may be considered now briefly and *seriatim*.

It has been known for a long time that ether is capable of producing such irritation to the respiratory passages as to cause laryngeal spasm, immoderate formation of saliva, "profuse secretion of thick tenacious mucus which hampers breathing," and occasionally inflammatory effects. In spite of this, one finds a most egregious instance of anachronism emanating from the Massachusetts General Hospital,[1] "where several hundred thoracoplasties have been carried out under ether anæsthesia, with exceptionally satisfactory results." Such a statement is all the more astounding when it is realized that virtually all other observers consider that, for operation on the tuberculous individual, the lungs should be at rest as much as possible, and that ether should be

avoided because of its irritant action on the lungs. Gordon,[2] discussing prophylaxis in post-operative pulmonary complications, says: "An irritant inhalation anæsthetic is to be avoided whenever possible, and if one is given, the anæsthesia should be smoothly and skilfully maintained. The reduction of the anæsthetic period to the shortest possible time is of definite prophylactic value. Every patient who presents himself must be *individualized* and the anæsthetic selected that is safest from every standpoint." Gilbert Brown,[3] states "that ether is definitely contra-indicated in patients who are suffering from pulmonary tuberculosis. The anæsthetist has at his disposal a number of drugs and a variety of methods of administering them." Need more be said at this time about the harm which may result from the use of irritant anæsthetics?

It is quite evident that we should refrain, as much as possible, from depressing the reflexes, for although secretions may be removed by aspiration, yet it is well that the protective mechanism of coughing be alert enough to respond to stimulation brought about by the accumulation of tracheobronchial secretions. With this in view, nitrous oxide, ethylene and cyclopropane will be found suitable for tuberculous patients as these drugs cause little or no irritation and do not interfere with the cough reflex for any length of time. The short-acting barbiturates may be used somewhat, considering the quiet induction and the rapid recovery they afford. However, it is characteristic of the various forms of regional anæsthesia that in none of them is respiratory effort increased, and in none is there reflex inhibition. Thus the danger of spreading the disease is reduced, and the expulsion of fluid is not hindered.

The penultimate principle for consideration is the disturbance which anæsthetics produce in metabolism. At once it may be said that all commonly used general anæsthetics give rise to some disturbance. Even cyclopropane causes an increase in blood sugar and an elevation in blood phosphorus;[4] and, too, a considerable diminution in serum potassium with a moderate lowering of the carbon dioxide combining power.[5] Ether anæsthesia always causes marked blood concentration.[6] This agent produces a true acido-

sis,[7, 8] which has been shown to be due in part to phosphoric acid[9, 10] from the muscles, and in part to lactic acid.[11] Although Beecher,[12] in his book, has slurred over the interesting subject of acid-base balance in anæsthesia, there are those who believe that the acidosis which anæsthetics produce may become a serious matter, particularly for the handicapped patient,[13] like the one presently under consideration. With regard to the effects of anæsthetics on the liver;[14] it has been demonstrated that the functions of this important and prodigious structure are definitely affected by ether,[15] in proportion to the degree and length of the narcosis; that with nitrous oxide, and with ethylene, when sufficient oxygen is supplied, anæsthesia produces neither immediate nor delayed impairment of hepatic function, but when the percentages of oxygen are purposely reduced, immediate impairment of function results and several days are required for recovery—what an important illustration of the danger of anoxia!—and, it has been found that cyclopropane anæsthesia does not seem to damage the liver under any circumstances.[16, 17] Analogous to the changes which take place in the liver are those which occur in the kidney.[18, 19, 20, 21] "It may be said that all general anæsthetics cause some depression of kidney activity, wherein the rate of secretion and composition of the urine are lowered; that the degree of depression varies directly with the depth of narcosis; and that the effects are influenced by the condition of the kidneys, by the water content of the blood, and by the duration of the anæsthesia."

This short account may suffice to draw attention to the ill-effects which anæsthetics may have on the blood, on the liver, and on the kidney, as they are brought on in the subject's fluid matrix, interrelated and interdependent, in similar fashion to the great interpenetration of the normal vital functions. They may serve as examples of the disturbance which anæsthetics are capable of producing in metabolism. They ought to point the way to selection in anæsthesia for the tuberculous individual. It is unbelievable that Beecher[22] should have failed utterly to have considered them. Leigh and I[5] have shown that, with the exception of blood dilution, these many disturbances which occur in general anæsthesia

do not appear in spinal anæsthesia. "Let it be remembered that some of these changes in metabolism may seriously impede the course of recovery in the patient who suffers some extensive debilitating lesion."[23]

The last requirement, that the method should permit an adequate provision of oxygen, needs little elucidation as the usefulness of oxygen in anæsthesia is well known and particularly in cases of pulmonary incapacity.

Of late years, in this Hospital, the Etherington-Wilson technique[24] of spinal anæthesia has been employed for rib resections in the performance of extrapleural thoracoplasty and the induction of extrapleural pneumothorax. Previous reports[25, 26, 27] have given good evidence of the usefulness of this kind of spinal anæsthesia for thoracic surgery. The practicability and the comparative value of this mode of anæsthesia are so convincing that reiteration of its efficacy in 291 instances seem justifiable.

The Etherington-Wilson technique depends on the fact that an anæsthetic solution of specific gravity 1.003 will rise at an even rate in the spinal fluid of specific gravity 1.007 while the patient is sitting. Four main zones have been established. Respectively, the doses and sitting-up times, suitable for these zones of analgesia, are shown in Table I.

TABLE I

Group	Doses of drug Percaine 1 : 1500 number of ml.	Time of bolt-upright posture in seconds	Areas at or below which operation may be performed
1	8—10	20—25	Poupart's ligament
2	10—12	30—35	Umbilicus
3	12—15	40—45	Nipple
4	12—15	45—60	Chest

"In practice the chosen quantity of percaine solution, 1 : 1500, warmed to the patient's temperature, is injected slowly, continuously, and evenly, while the patient *sits as erect as possible*. The injection should take from 20 to 25 seconds. The *total time* should be watched very accurately and *must be from the beginning of the injection to the end of the sitting up*. At the end of the allotted number of

seconds the upright position is quickly changed to that of recumbency and the head of the table is tilted slightly downwards."

Perhaps the most important part of the preparation of a patient for regional anæsthesia is adequate sedation. We have made a habit of giving ¼ gr. of morphine with 1/100 gr. scopolamine 90 minutes before the time of operation and repeating these 60 minutes later if the patient is not thoroughly well bemuddled and languorous. But the doses should not be fixed for all cases. It is better to consider each case independently. Thus the subject ought to be in a state of complete abandonment on being taken to the operating room for the spinal method of regional anæsthesia which has been set forth just now.

So soon as the patient is in position for operation, a glucose-saline solution is given intravenously, and kept up throughout the performance. This solution may be replaced by blood or plasma readily. To the fluid, within the intravenous tube, may be added momentarily either resuscitating or sedative drugs.

Concerning the use of analeptics, I am strongly of the opinion that they should not be given routinely. They are apt to stimulate the central nervous system and to disturb the effects of sedatives. After Melville[28] discovered the remarkably synergistic and analeptic action of the mixture of the pressor principle of posterior pituitary extract with ephedrine, this mixture was first used in clinical anæsthesia in Montreal (1937).[13] In this hospital we have administered these two drugs in 100 instances of the 291 operations mentioned, or in 34 per cent of cases. " In the early days we used them more frequently—over 50 per cent; but, latterly, they seem to be needed not more often than in 20 per cent. The criterion for the employment of this analeptic mixture is a marked fall in blood pressure. These figures should cause those who give analeptic drugs routinely to change their practice and thoughtfully to avoid undoing, at least in part, that which was deliberately done with good reason, namely, the dissipation of fear. Again, the fact than analeptics were not needed in so many of these cases tends to prove the statement made before in favour of

Figure 1.

GRACE DART HOME HOSPITAL
MONTREAL

ANAESTHESIA RECORD

No. Ward Date Aug. 6th. 1941.
Name J—, F M°. age: 31. years.
Surgeon F. B. G. Anaesthetist W. B. & M. B. L.
Operation Thoracoplasty 3rd: Right ribs 7 & 8.
Special physical findings

Preoperative drugs Morphine gr ¼ & Scopolamine gr 1/100 at 11:30 a.m. & 1:15 p.m.
Anaesthetics Perrains 12 ml. 1/500 Method Spinal, Elk. Wilson Apparatus Face-Sok.
55 secs

Hour	p. m.											
	1:45	15	30	45		15	30	45		15	30	45

Q pr nares and C.S. i.v. continued.
Rx drug s immediately.
Strychnine gr 1/30 s.c. b.i.d. X3
Recovery routine Ether C

POSTOPERATIVE: Condition and treatment

W.B.

Figure 2

GRACE DART HOME HOSPITAL

ANAESTHESIA RECORD

No. Ward Date 21 11 41

Name R — Miss W. Age 30 years

Surgeon F.B.G. Anaesthetists M.D.L. + W.B.

Operation Thoracoplasty - 1st. : Left ribs 3, 2 + 1.

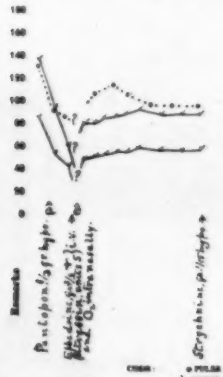
Special physical findings

Preoperative drugs Morphine gr. 1/6 + Scopolamine c.c. gr. 1/150 at 12.30 p.m. + gr. 1/150 at 1.30.

Anaesthetics Pentothal 1/20 Methedrine, Ether, Wilson Apparatus Luer-Yale.

55 secs

Hour: a.m. p.m. 2 15 30 45 3 15 30 45 15 30 45



Glucose 5% infusion I.V. O₂ continued.
Ephedrine gr. 1/6 + Pituitrin 5 units hypo.
Strychnine gr. 1/30 hypo. q. 4 h. x 3.
Uneventful recovery.

POSTOPERATIVE: Condition and treatment

W.B.

complete sedation, particularly 'that there is much less shock, much less circulatory disturbance, than there used to be, and hardly ever any nausea.'"

Figures 1 and 2, with their legends, require little further explanations. They are borrowed from a paper entitled, "Spinal Anæsthesia for Thoracic Surgery."[27] From the former, it will be seen that in this case the mixture of ephedrine and pituitary extract was not used. In the case of the latter figure it is evident that the administration of the analeptic mixture was followed by a prompt amelioration of the patient's condition. It may be said that, of the pituitary extract preparations on the market, that known as ***pitressin*** has the greatest amount of pressor principle.

The plan has been adopted of giving oxygen intrapharyngeally from the beginning of anæsthesia throughout well into the early post-operative period. This is done because with this type of spinal anæsthesia, there is always some respiratory depression, the blood pressure falls, the pulse slows, and blood dilution takes place; in consequence, the oxygen carrying power of the blood is lowered. In this connexion, in case of acute collapse, it is imperative that the anæsthetist be always prepared to do artificial respiration by the method of Guedel, that is, to administer oxygen, interruptedly and rhythmically, directly at the bifurcation of the trachea through an intratracheal tube.

TABLE II

Particulars	Spinal anæsthesia number	Other anæsthesias number
Number of individuals	105	29
Number of operations	291	65
Causes of death:		
Anæsthesia	4	0
Hæmoptisis (massive)	1	3
Hæmorrhage (wound)	1	0
Pneumonia	0	3
Pericarditis (Tbc.)	1	0
Septicæmia (wound infection) ...	1	0
Spread of Tbc. (under 3 months)	1	2

Table II shows that there were four deaths with anæsthesia as the ascribed cause, that is, 1.34 per cent of operations and 3.77 per cent of individuals. "Two of these occurred rather early in the series, and at once we hasten to say that neither of these patients ought to have been operated upon. Each was so ill and so debilitated that virtually they were *in extremis*. They died shortly after the anæsthetic had been administered. At the time we had not been prepared for the better methods of resuscitation previously mentioned, namely, we had not yet adopted the plan of promptly starting an intravenous injection of fluid, and we had not yet had ready the equipment for artificial intratracheal respiration. The details of the third and fourth deaths have been reported elsewhere.[27] Suffice it to say here of the former, that following the anæsthesia there were convulsions off and on for 18 hours and then he died. It was found difficult to offer an explanation.

The fourth case stopped breathing at the end of the operation and within a few seconds there seemed to be no heart beat. After strenuous resuscitative measures, circulation and respiration were re-established, but breathing was shallow and there were spasms of the arms and neck which lasted for a few seconds and occurred about every half hour. Sometimes these were accompanied by crying or screaming. The spasms increased, she did not regain consciousness and died on the third day after operation. It would seem that in this patient the blood supply to the brain was cut off too long for consciousness to return. In this case too, we had not been prepared for the better methods of resuscitation mentioned above.

Summary

1. Some of the effects of anæsthetics have been reviewed with regard to the production of irritation and damage to the lung, to their influence on the protective reflexes, and to metabolic activity.

2. It has been pointed out that ether may do harm to the lungs directly, and may interfere seriously with the tuberculous patient through its disturbances in metabolism.

3. Regional anæsthesia is advocated for the tuberculous

patient because the many injurious actions, which general anæsthesia is capable of producing, do not take place in any of its various forms.

4. An abundant use of oxygen is advocated in surgical procedures for the handicapped patient, especially the tuberculous patient.

5. The place of analeptics has been made clear.

6. Supporting fluids have been considered.

7. The Etherington-Wilson technique of spinal anæsthesia is advised for major surgical procedures, at least, and for the following reasons:

(*a*) As breathing is so quiet and as there is so little bleeding, the surgeon can do his work with ease and expedition.

(*b*) Paradoxical breathing is seldom excessive in the chest cases.

(*c*) Although seemingly imminent, post-operative hæmorrhage rarely occurs to any extent.

(*d*) During anæsthesia and also, after operation, secretions in the air passages are at a minimum.

(*e*) And the patient is relatively more comfortable in the post-operative period after this type of anæsthesia than in that after others, owing to the lasting action of nupercaine.

Finally, *assurance is given of the excellent recovery following* "high" spinal anæsthesia by the "sitting-up" method. It has been said[27] that "the best method is not necessarily the least dangerous, but rather that which offers an *optimum* for restoration. Undoubtedly, the good results are in large measure due to the fact that, with the exception of blood dilution, the many changes which are apt to take place in metabolism from general anæsthesia do not appear in spinal anæsthesia."

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TWO CASES OF CONVULSIONS

BY

F. F. WADDY, M.B.

SOME time ago I was consulted about the following case after death had taken place:

Operation for Webbed Fingers on a Child Aged Two Years.

When the mask was removed at the conclusion of the operation respiration had ceased, the pulse was not palpable, the colour of the child was fairly good. This was about 6. p.m.

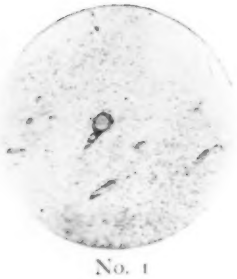
Resuscitation was attempted as follows:

Coramine 1 c.c. was given.
Artificial respiration with oxygen applied.

Endotracheal catheter passed—oxygen given through it, and later carbon dioxide and oxygen. Adrenalin 1 c.c. into the heart. No pulse present still. Abdomen slit—cardiac massage through the diaphragm. Heart muscle flabby. Heart started. Artificial respiration continued for a few minutes before the child took a breath properly—the colour and the pulse improved. Pupils were still dilated. The abdomen was closed. Endotracheal catheter was left *in situ*, and the child was returned to the ward after about 30 minutes' time, when the condition improved.

During the night the child's condition deteriorated—then came twitchings of the face and arms and the child was very restless. Nepenthe M ii. given, oxygen was administered, and coramine ½ c.c. four hourly. There was some improvement for a time, then the condition again deteriorated. About 9 a.m. there was cyanosis and twitching of the face and arms. The child was put into an oxygen tent. The child died at 10 a.m.

I immediately communicated with the pathologist who would conduct the post-mortem, and asked him to keep the



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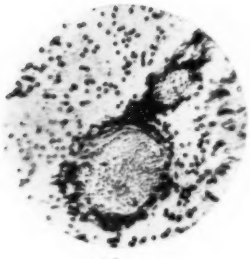

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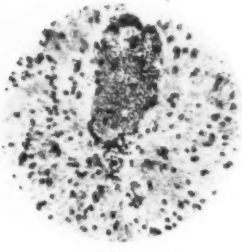

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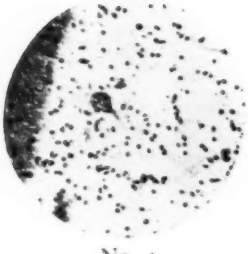

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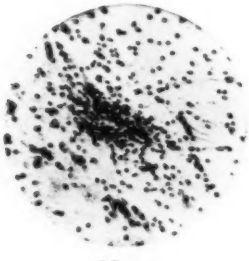

No. 5

F.F.W.

brain. The post-mortem examination was carried out that afternoon.

There was no gross physical lesion elsewhere. The thymus was slightly overweight. There was a yellowish discoloration of part of the cerebral cortex and the caudate nucleus. Such a discoloration is noted in a few of the cases where brains have been examined after death from anoxia. Abbott and Courville[1] note it in a case of bilateral necrosis of the globus pallidus. There was a large number of sections made, but in my ignorance of histological procedure I had failed to guard against cortical autolysis so that in all my sections simple evidence of necrosis cannot be regarded as sure evidence of ante-mortem damage.

May I say here that if an anæsthetist should get the opportunity of seizing a case like this he should remember to put the body in a refrigerator immediately after death, or pack ice all round the skull?

Fortunately, however, there is evidence of ante-mortem damage. Nearly every photograph shows an enormous engorgement of all the blood vessels.

Photograph No. 1. Nissl. 2/3 objective.

Shows two engorged vessels in the cerebral cortex with a large number of white cells between them.

Photograph No. 2. Nissl. 1/6 objective.

This is the previous slide under a high power objective showing that the cells lying between the two blood vessels are white cells. The whole slide is not in focus as it is slightly uneven.

Photograph No. 3. H. and V. G. 2/3 objective. BASAL GANGLIA.

Shows an enormously engorged vessel with perivascular infiltration.

Photograph No. 4. Nissl. 1/6 objective. MID BRAIN.

Shows a motor nerve cell with disappearance of dendrites, "powdering" of Nissl. granules and displacement of the nucleus to the side of the cell. There is considerable swelling of the axon and early satellitosis. This is not an irrecoverable condition, but undoubtedly represents at least a temporary loss of function.

Photograph No. 5. Nissl. 2/3 objective. MEDULLA.

Shows intensive clumping of satellite cells around the remains of (two?) nerve cells.

1/6 objective. Ditto.

Discussion of Histological Evidence

The engorgement of the vessels, I think, can be taken as an indication of a pathological process in the brain, and I do not think it should be confused with an engorgement at the time of asphyxia or of a reactionary engorgement immediately following asphyxia, as this is the state of the brain 16 hours afterwards.

There is evidence in several slides of slight perivascular infiltration. The two slides shown, comprising Photographs 1 to 3 are outstanding examples. It is interesting to notice that this is seen in cases of carbon monoxide poisoning, which is merely a specialized case of anoxia.

In arguing that local stagnation is the important factor which designates the local area of necrosis Courville[2] points out that necrosed areas are usually around the vessels. The collection of white cells around these vessels may be significant.

Secondly, in the mid brain there is evidence of what might be termed "a very sick motor cell" which does not point to more than a loss of function but taken in conjunction with the other evidence it seems likely that this cell is dying. There are several cells with swollen axons—this is but one example.

In the third slide there is an intensive clumping of satellite cells, and there is here a remaining part of one, if not two motor cells. There is evidence of satellitosis in several other sections but this one shown is the only one to which an impartial mind can attach certain pathological importance.

From a histological point of view this may not be weighty evidence, but I submit that when one views all these things as a whole, and remembers that the child only survived 16 hours after the fatal incident that it is remarkable that so much evidence of tissue damage in the central nervous system can be found, especially when it is remembered what a power of recovery the tissues in the young have. Furthermore, although some 30 sections were stained the chances of missing the level of a group of satellite cells like that shown in Photograph 3 must be extremely high.

If, therefore, one grants that definite damage to the nerve cells has occurred it remains to find out from what cause.

There are two obvious causes:

1. The ether damaged these and other cells, and so caused the original cessation of respiration and the later sequelæ.

2. The ether caused the cessation of respiration, etc., and the consequent anoxia did the permanent damage.

Had this been the first reported case of its kind a considerable time might be spent in arguing it, but in view of Courville's work and his comparison with deaths due to asphyxia in his series, and those due to anaemia in Gildea and Cobb's series, I think it can be accepted that the second explanation is the correct one.

There are the following points of special interest:

1. This is the first case so far as I know of nerve cell damage to be reported as low in the central nervous system as the basal ganglia and medulla from anoxia. All cases up to date have dealt with the cortex and caudate and lenticular nuclei.

2. As far as I am aware there is no such report in existence of a death which has followed ether anæsthesia. Courville[3] recently wrote a paper on "Ether Anæsthesia and Cerebral Anoxia," in which he quotes only two cases: one alive with a decerebrate rigidity and blindness, and one in which an autopsy was performed without examination of the brain. He says "as yet the detailed cerebral changes after anoxæmia under ether anæsthesia (actually due to cardiac or respiratory failure) are unknown." This is one such case.

3. From the point of view of discussion on convulsions under anæsthesia it will be noted that there were no convulsions during anæsthesia, but twitchings of the face and arms started about 1 A.M.—seven hours later. At this time nearly all the ether administered must have been eliminated but the effects of the anoxia remained.

Conclusions

It seems to me fair to say that while the histological evidence, if taken piece-meal, must be considered inconclusive, the whole picture certainly points to nerve cell damage from anoxia and that the history of the case is almost conclusive. It seems probable that had the patient lived a little longer

and had steps been taken to fix the brain immediately, that there would have been abundant certain evidence of this.

My thanks are due to Dr. Hilda M. Dean for the notes on this case, and for the post-operative treatment of which she was responsible.

I have to thank Dr. E. S. Duthie for securing the specimen for me, Dr. Ruby Stern for her willing help with, and instruction on, the histological appearances, and Mr. Ashford who made the photo-micrographs.

The second case that I wish to record is one in which convulsions followed a loss of blood.

I was asked to see a patient, aged 75, with a view to giving him an anæsthetic for a per-urethral resection of prostate. His general condition was poor. Myocardium poor. Lungs clear. B.P. 148/92. He was a well-educated man, who stated that he had an idiosyncrasy to morphine. In view of this no preliminary sedative was given, but ephedrine gr. 1 was given ten minutes before operation. At 3.37 p.m. he was given 10 c.c. 1/1500 percaine intrathecally. It is my practice not to use a perineal anæsthesia but to get analgesia as high as the umbilicus in case a suprapubic cystotomy should be deemed advisable. This obviates the necessity of having, for any reason, to supplement the original anæsthetic. At 3.50 the operation was commenced, ½ c.c. veritol having been given intramuscularly at 3.45. At 4.30 p.m. the patient was looking a little pale and a further ½ c.c. of veritol was given intramuscularly. At this time the surgeon told me that he had "just about finished." A few minutes later the surgeon warned me in a whisper that the last cut which he had intended to make with the diathermy had apparently cut across a large vessel. At this stage the patient said he was very comfortable but "a little tired." In spite of copious washing with hot lotion through the endoscope it was impossible to allay the bleeding sufficiently to see a bleeding point which could be treated with a diathermy. The bleeding was profuse and it was decided to do a suprapubic cystotomy immediately. The patient was put in position, and the towels had been put on when, just before the surgeon made the incision, the patient had what obviously

would have been a generalized convulsion if his legs had not been paralysed. His eyes twitched, his jaw made chewing movements consisting chiefly of clonic masseter contractions which were so violent that the nurse whom I had instructed to hold the jaw was unable to keep her fingers on it. The hands and arms moved violently in clonic contractions. Before this occurred I had put some coramine in a syringe, and now asked the surgeon to proceed immediately to secure the bleeding point. As I pulled out the arm, the patient gave a last shudder and became flaccid. The eyeballs rotated upwards and the pupils became fully dilated, not reacting to light. There was no corneal reflex. The pulse could not be felt, respiration ceased. I injected ½ c.c. of coramine into a large vein in the arm, told the nurse to hold the arm up in the hope that the coramine would be returned to the heart, and gave the patient's chest a few rhythmic squeezes. In the meantime the surgeon had opened the bladder which contained well over a pint of blood and had packed gauze firmly in the prostatic cavity. The artificial respiration restored normal respiration and the pulse and eye reflexes returned. The bleeding point was secured and the operation completed without further incident, evcept that as the patient partially recovered consciousness he vomited.

The patient did not become fully conscious until 8.20 p.m., and had no precise memory of the operation—remembering only being given the spinal anæsthetic and lying on the table for a while. His recovery was not rapid, but was complete, although it was jeopardized by hiccup which lasted for five days.

The convulsion was a typical asphyxial convulsion, the like of which I have only seen when "pushing" nitrous oxide in an alcoholic, when I unintentionally employed the secondary saturation technique. Extensor spasms of a clonic type were followed by a temporary cardiac and respiratory failure.

Cerebral anoxia was undoubtedly produced by the sudden reduction in the quantity of circulating blood in addition to a previous comparative anoxia and was probably only combated by the combined effect of the 4.30 intramuscular dose of veritol, together with the 4.45 intravenous dose

of coramine which, presumably, together raised the blood pressure sufficiently to allow the cerebral circulation to recover.

It will be noticed that the convulsions were of "central" origin as they only reached down to the level of the spinal anæsthesia.

Barcroft, in 1920, divided anoxæmia, or more correctly, anoxia, into three types:

1. The anoxic. 2. The anæmic. 3. The stagnant.

In the first class Barcroft includes normal respiration in an atmosphere deficient in oxygen, and normal respiration where the atmosphere has not normal access to the blood, as in lung disease. I should think he would also have added reduced respiration as one sees it in spinal anæsthesia which results in insufficient oxygen being admitted to the blood, although there is here something of the third, or stagnant, type.

In his third or stagnant type he includes blood supplied to the tissues in quantities insufficient to allow of adequate oxygenation as in shock, lowered blood pressure and haemorrhage.

In my second case hæmorrhage followed insufficient ventilation of the lungs and resulted in a very violent convulsion which to my mind is typical of anoxia. Why the average case of hæmorrhage without anæsthesia usually results in fainting or loss of consciousness without convulsions is a difficult question, but it is possibly due to the fact that the tissues normally have a reasonable reserve of oxygen.

The only case comparable to this that I can quote is one in which Batten and Courville described mental disturbance in a patient following a profuse hæmorrhage while under nitrous oxide anæsthesia.[4] Only slight cyanosis was noticed at the time, and convulsions followed 15 minutes later.

Beecher[5] quotes Gildea and Cobbs' observations on convulsions due to cerebral anæmia and says "experienced anæsthetists who have had the misfortune to witness 'ether convulsions' will recognize that Gildea and Cobb have given unintentionally a remarkable description of this cata-

strophe, and possibly the explanation of the cause of it as well."

In his earlier works Courville was inclined to stress the fact that the damage due to anoxia was in the presence of nitrous oxide, although later he admits that the effect is purely one of anoxia.[6]

To summarize we have:

1. Numerous reported cases of so-called "ether convulsions"—i.e. convulsions occurring during ether anæsthesia, in many of which the patient's colour is referred to as "poor."
2. Convulsions occurring due to cerebral anæmia as reported by Gildea and Cobb.
3. Convulsions occurring during nitrous oxide anæsthesia and resulting in permanent cerebral tissue damage.
4. Convulsions occurring long after ether has been withdrawn, but after damage has been inflicted on the tissues of the central nervous system, as in my first case.
5. Convulsions occurring after hæmorrhage where no ether or other general anæsthetic has been used, as in my second case.

The only explanation that I can see that fits all these cases is that the convulsions are due to anoxia and that permanent damage due to anoxia has been proved in cases where the victim has survived sufficiently long for its effects to be established.

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ANÆSTHETIC RECORDING AS PRACTISED AT MADISON

BY

E. M. CHIVERS, D.A.

[Much valuable clinical material is wasted through lack of any but the barest recording of all the facts concerning patients, before, during and after anæsthesia. We hope the following article may help those who desire to remedy this state of affairs.—Ed.]

THE system of recording anæsthetic data now in use at the Wisconsin General Hospital, Madison, and in many other American institutions is based on the Hollerith Punch Card. This system has been used in workshops and business houses for many years, but is quite novel in relation to anæsthesia. It has the advantage of being sufficiently adaptable to permit the recording of all salient facts relating to a patient, and the use of a numerical code for this purpose reduces the space required to a minimum.

Some form of recording is necessary for statistical purposes, besides being of personal interest to the anæsthetist concerned; it can be done simply with pencil and paper, or by some mechanical means such as the punch card system—the latter being of great advantage when large series of cases have to be analysed as may happen in a research institution. An additional advantage is the fact that after the card has been punched a permanent record is always available for future reference.

HISTORY

In the year 1932 a recording system was introduced into the Anæsthetic Department of the University of Wisconsin General Hospital. Year by year this system has been modified and improved until the present comprehensive system has resulted.

The idea originated from a business friend on a visit to Dr. Ralph Waters at the time when the resident anæsthetist

was completing the arduous task of sorting data on a series of 1500 spinal anæsthetics. He was appalled at the amount of work involved, and suggested the use of the punch card system in this type of work. Dr. Waters welcomed the suggestion, recognised the advantages and possibilities, and was able to modify it to meet the needs of the department.

For about five years experiments were made with various codes and types of cards until in 1937 the American Society of Anæsthetists officially recognised the system, and appointed a committee, with Dr. Meyer Saklad, of Providence, as chairman, to investigate the subject and frame a suitable standard code for general use in the U.S.A., thus eliminating the personal factor as far as possible. This Committee published a booklet "How to Code," which defined the terms to be used and set the standard for recording various complications (i.e. how severe a cough should be before it was recorded).

The present code has been used since 1940, it will be reviewed in 1945 with a view to adopting any useful suggestions for improvement that have been made in the interim.

Recording

The actual recording begins when the operation lists for the day are posted up in the theatre. The anæsthetist, house surgeon, students and any interested visitors start on a "premedication round," during which patients are examined fully, and any special investigations are ordered or done, the facts are recorded as concisely as possible on the back of the anæsthetic record chart.

Suitable premedication is ordered, and it is the duty of the anæsthetist to ascertain that this is administered. The charts are taken to the theatre, and any relevant facts filled in during and after operation. Thence the charts go to the secretary's office, where each is issued with a serial number which, with the patient's name, is entered in a card index. This facilitates any future reference to a case. It is the duty of the anæsthetist to visit the patient at intervals after operation until all "anæsthetic sequelæ" are past, and record post-operative data. Should the patient die, a special death chart, giving full details is made out, and at the weekly staff

meeting the case is discussed, and it is decided whether the anæsthetic in any way contributed to the fatality, and what cause of death should be entered for statistical purposes. All completed anæsthetic charts are taken to the meeting, where they are used for reference or to illustrate any special points to students. After the meeting the charts are returned to their authors for coding.

Coding

The cards used measure $3\frac{1}{4}$ in. by $7\frac{3}{8}$ in. Each card is divided into eighty vertical columns of digits from 0 to 9. (Fig. 1.)

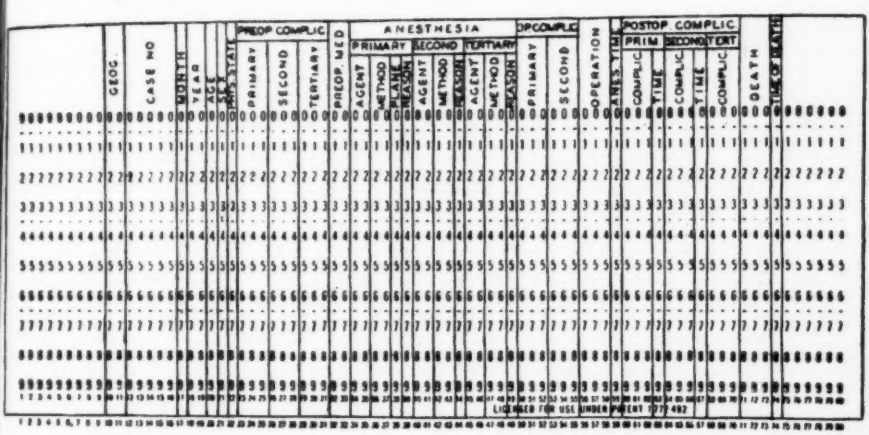


Fig. 1

The card is also divided into fields. Each field may consist of any number of columns depending on the number of variables that have to be considered in that field.

To record the months of the year one column is sufficient, as twelve positions can be coded by adding two extra positions (x and y) above 0. When more than one column is allotted to a field the extra positions (x and y) are not used as in the case of premedication, where in a field consisting of two columns 99 variables can be coded or 999 variables in the three column field allotted to operation. On the card that

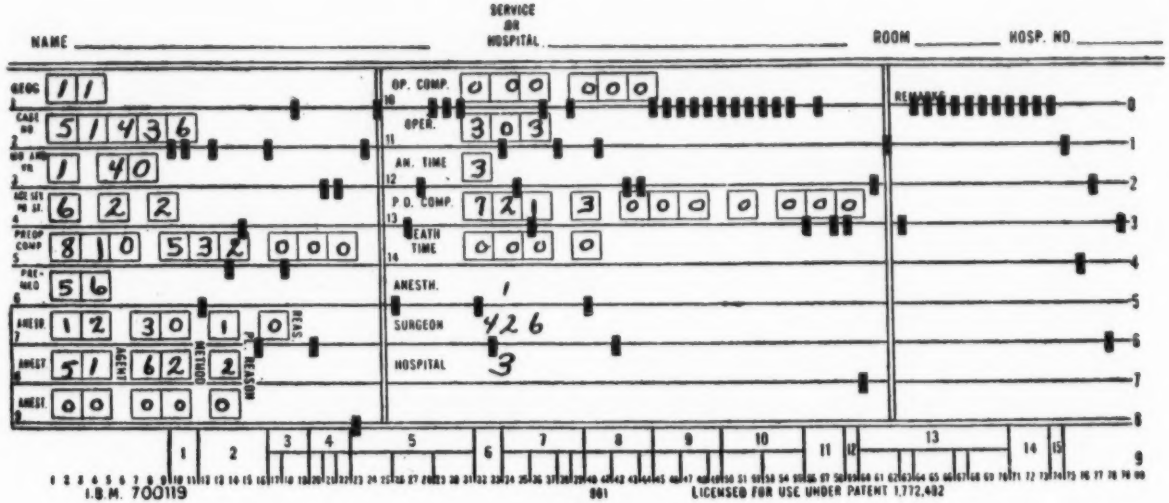

NAME
SERVICE OR HOSPITAL
ROOM
HOSP. NO.
GEOG
CASE NO
AGE YR
AGE MO OR D.
PREOP COMP
PRE-MED
ANESTH
ANEST
ANEST
REGS
METHOD
AGENT
PT. REASON
OP. COMP.
OPER.
AN. TIME
P.O. COMP
DEATH TIME
ANESTH.
SURGEON
HOSPITAL
REMARKS
I.B.M. 700119
001
LICENSED FOR USE UNDER PATENT 1,772,492

FIG. 2

is ready for coding the 80 vertical columns are not marked, but represented by the small figures printed at the bottom of the card, with the numbers representing the fields immediately above them. The resulting blank card is divided into horizontal columns containing "boxes" or small squares, in which the code figures are written. (Fig. 2.)

The arrangement of the boxes permits the later stage of punching to be done directly from the card. *The General code,* which is used only for columns 10 to 74 on the card, is very comprehensive, and consists of two big divisions—Operations and Complications. Examples of general code are given below.

The committee decided that columns 1 to 9 should be left open and used as a private code. Each institution is encouraged to make its own to suit the particular phenomena in which it is interested.

Columns 75 and 76 are devoted to the anæsthetist; columns 77 and 78 to the surgeon, so each institution has to make its own code for these columns, and in each case it is possible to code 99 individuals. The committee was also responsible for assigning a distinguishing or geographical number to each institution using the recording system; this is coded in columns 10 and 11. *The Private Code* columns are written down first on the extreme right of the card. (Fig. 4.) Then starting at the top left-hand corner columns 10 to 74 are coded, using the general code. In box 1, corresponding to columns 10 and 11, or field 1, the geographical number is coded; below in box 2, corresponding to field 2, or columns 12 to 16, the case number; and so on until the card is completed. Column 80 is not used, but left open and used as one method of check before sorting.

WISCONSIN GENERAL HOSPITAL PRIVATE CODE

Conduct of Anæsthesia (Column 1)

0 Satisfactory
1 Unsatisfactory relaxation
2 Other surgical dissatisfaction
3 Obstruction—oxygen want
4 Unsatisfactory—other causes
5 Unsatisfactory to patient
(Remaining numbers unused)

Induction Complications (Column 2)

0 None
1 Apprehension—marked
2 Excitement—moderate
3 Excitement—severe
4 Coughing
5 Proc. resc. with convulsions
6 Proc. resc. without convulsions
7 Convulsions with other agents
8 Slow
9 Others

Time from Anæsthesia to Operation (Induction time—Column 13)

0 None
1 Less than 5 minutes
2 5—10 minutes
3 10—15 minutes
4 15—20 minutes
5 20—25 minutes
6 25—30 minutes
7 30—45 minutes
8 45—60 minutes
9 More than 1 hour

Treatment of the Table (Column 4)

0 None
1 Intravenous fluids
2 Subcutaneous fluids
3 Opiates
4 Oxygen therapy
5 Transfusion
6 Tracheobronchial toilet
7 Rectal fluids
8 Barbiturates
9 CO_2 inhalations
x Stomach tube
y Anæsthesia mixture diluted with air

Endotracheal Route (Intubation)

0 None
1 Oral with cuff
2 Oral with pack
3 Oral airway (under mask)
4 Nasal with cuff or pack, by vision
5 Nasal with cuff or pack, blind
6 Nasal without cuff or pack, by vision
7 Nasal without cuff or pack, blind
8 Nasal airway (under mask), blind
9 Nasal airway (under mask), blind
x Oral without cuff or pack, connected open
y Blind oral.

There are now two columns devoted to position on the table (Nos. 6 and 7). This is done so that one can code two things simultaneously, e.g. lithotomy position followed by Trendelenburg.

Position (Column 6)

0 Supine
1 Prone
2 Lateral
3 Lithotomy
4 Lithotomy—Trendelenburg
5 Fowlers moderate
6 Fowlers extreme
7 Upright
8 Trendelenburg—moderate
9 Trendelenburg—extreme

Position (Column 7)

0 None
1 Tourniquet
2 Rest
3 Supine
4 Prone
5 Lateral
6 Trendelenburg
7 Lithotomy
8 Upright

Column 9 we devote to "special file." The object is to record points of interest, so that the records concerned can be got at easily if needed.

Special File (Column 9)

0 None
1 Respiratory
2 Circulatory
3 Atypical signs
4 Atypical technique
5
6 Others

Recovery (Complications in Theatre) (Column 8)

0 None (Phar. ref. absent)
1 Pharyng. ref. present
2 Retching
3 Vomiting
4 Mild excitement
5 Severe excitement
6 Resp. obstr. severe with reflexes
7 Resp. obstr. severe without reflexes

Column 79 we use to record post-operative treatment under the following headings. (In this, and in columns 1 and 4 we allow several figures to be punched if necessary, but nowhere else on the card.)

Post-operative Treatment (Column 79)

0 None
1 Intravenous fluids
2 Subcutaneous fluids
3 Opiates
4 Oxygen therapy
5 Transfusion
6 Tracheobronchial toilet
7 Rectal fluids
8 Barbiturates
9 CO_2 inhalations
x Stomach tube passed

EXAMPLES OF GENERAL CODE

Premedication
(Columns 32 and 33)

00 None
01 Atropine
02 Scopolamine
03 Barbiturate
04 Codein
05 Dilaudid
06 Morphine
07 Omnopon
08 Heroin
09 Alcohol
11 Atropine-scopolamine
13 ,, barbiturate
14 ,, codein
15 ,, dilaudid
16 ,, morphine
17 ,, omnopon
18 ,, heroin
19 ,, alcohol
23 Scopolamine-barbiturate
24 ,, codein
25 ,, dilaudid
26 ,, morphine
27 ,, omnopon
28 ,, heroin
29 ,, alcohol
34 Barbiturate-codein
35 ,, dilaudid
36 ,, morphine
37 ,, omnopon
38 ,, heroin
39 ,, alcohol
43 Atropine-scopolamine-barbiturate
44 ,, ,, codein
45 ,, ,, dilaudid
46 ,, ,, morphine
47 ,, ,, omnopon
48 ,, ,, heroin
54 Barbiturate-scopolamine-codein
55 ,, ,, dilaudid
56 ,, ,, morphine
57 ,, ,, omnopon
58 ,, ,, heroin
64 Barbiturate-atropine-codein
65 ,, ,, dilaudid
66 ,, ,, morphine
67 ,, ,, omnopon
68 ,, ,, heroin
99 Others

Anæsthesia Time
(Column 59)

0 No anæsthesia
1 Less than 5 minutes
2 ,, ,, 30 ,,
3 ,, ,, 60 ,,
4 ,, ,, 90 ,,
5 ,, ,, 2 hours
6 ,, ,, 3 ,,
7 ,, ,, 4 ,,
8 ,, ,, 5 ,,
9 Over 5 hours

Agents

00 None
10 Carbon dioxide
11 Chloroform
12 Cyclopropane
13 Divinyl ether
14 Ethyl chloride
15 Diethyl ether
16 Ethylene-air
17 Ethylene-oxygen
18 Nitrous oxide-air
19 Nitrous oxide-oxygen
20 Somnoform
29 Others, inhalation

30 Alcohol
31 Evipan
32 Pentothal
39 Other barbiturates

40 Cocaine
41 Metycaine
42 Percaine
43 ,, 1:200
44 ,, 1:1500
45 ,, -procaine
46 Pantocain
47 ,, 1% saline
48 ,, procaine
49 ,, glucose
50 ,, —light
51 Procaine
52 ,, alcohol
53 ,, glucose
54 ,, light
59 Others, regional

60 Paraldehyde
61 Avertin
62 Ether-oil
63 Tri-chlor-ethanol

70 Helium-oxygen
71 Nitrogen
72 Nitrogen-CO_2
79 Others, therapeutic

99 Others

Methods

00 None
01 Open mask
02 Open with tube under mask (endo.)
03 Semi-open (mask covered with towels)
04 Semi-open with endo. tube
05 Pharyngeal insufflation
06 Insufflation endotracheal
07 Closed: no absorption (Clover, etc.)

10 Semi-closed (Boyle, McKesson, etc.)
11 Semi-closed with positive pressure
12 ,, endopharyngeal
13 ,, endopharyngeal, pos. press.
14 ,, endotracheal
15 ,, endotracheal, pos. press.
17 ,, endobronchial
18 ,, endobronchial, pos. press.

20 Absorption to and fro
21 ,, ,, pos. press.
22 ,, ,, controlled resp.
23 ,, ,, endopharyngeal
24 ,, ,, endotracheal
26 ,, ,, endo. cont. resp.
27 ,, ,, endobronchial
28 ,, ,, endobr. pos. press.
29 ,, ,, endobr. cont. resp.

30 Absorption circle filter
31 ,, ,, pos. pres.
32 ,, ,, cont. resp.
33 ,, ,, endopharyngeal
34 ,, ,, endotracheal
35 ,, ,, endotracheal pos. press.
36 ,, ,, endotracheal cont. resp.
37 ,, ,, endobronchial
38 ,, ,, endobronchial pos. press.
39 ,, ,, endobronchial cont. resp.

40 Intravenous
50 Rectal
60 Topical application
61 Infiltration
62 Field block
63 N.B. cervical plexus
64 ,, brachial plexus
65 ,, intercostal thoracic
66 ,, lumbar
67 ,, thoraco-lumbar combined
68 Splanchnic block
69 Miscellaneous nerve block
70 Sacral block
71 Caudal block
72 Sacral and caudal (trans-sacral)
73 Epidural
80 Spinal, no adrenergic drug
81 Spinal with ephedrine
82 Spinal with other adren. drug
83 Serial spinal
99 Other methods

OPERATIONS

Head and Neck

Head—Superficial
100 Suture lacer, I & D abscess
101 Scalp and face operations
109 Others, head, superficial

Intracranial
110 Ventriculogram
111 Section nerve—ligat. vessel
112 Decomp. elev. fracture
113 Explor. removal brain tumor
114 Drainage, brain abscess
115 Encephalogram
119 Others, intracranial

Neck
120 Suture lacer., I & D superf. abscess
121 Excis. superf. glands
122 Oesophag. diverticula, upper
123 I & D deep abscess, neck
124 Tracheotomy
125 Cervical sympathectomy
126 Laryngeotomy (laryngectomy)
127 Branch. thyroglossal cysts
128 Ligat. jugular: deep gland dissect.
129 Others, neck

Thyroid and parathyroid
130 Thyroidectomy—first stage (Hemi.)
131 ,, later stages
132 ,, both lobes
133 ,, ablation
134 Excision adenoma
135 Subtotal hemithyroid, complete
136 Ligation, thyroid artery
137 Para-thyroidectomy
139 Others, thyroid

Ear, nose and throat
140 Ear, superficial operations
141 Plastics on nose, external
142 Operations on lip
143 Insertion Ra.
144 Combination superficial plastics
145 Nerve anastomoses, face and neck
146
150 Mastoidectomy, cortical
151 ,, radical
152 Intra-nasal operations
153 Caldwell-Luc
154 Palate operations
155 Jaw operations
156 Glossectomy
157 Parotid operations
159 Biopsy tongue, mouth
160 Laryngoscopy: bronchosc., vocal cord opns.
161 Oesophagoscopy, gastroscopy
162 Tonsillectomy
169 Nasopharyngeal operations, misc.
170 I and D retro-pharyngeal abscess
171 ,, sublingual abscess
172 ,, peri-tonsillar abscess
179 Other peri-oral operations

Dental
180 Extraction, simple
181 Extraction, impacted
182 Wiring fractured jaw
183 Complete extraction
184 Dental cysts and tumours
185 Dental abscesses
186 Others, dental

Eye
190 Lids, superficial, F.B., plastics
191 Lachrimal duct or gland operations
192 Eye muscles, cornea
193 Iridectomy, cataract, deep F.B.
194 Enucleation
195 Exenteration of orbit
199 Others, eye

Thorax

Superficial
200 I & D abscesses, lacer., dressings
201 Excision of breast tumour
202 Local excision, breast
203 Radical excision of breast
209 Others, superficial

Extrapleural operations
210 Operations on thoracic cage
211 Thoracoplasty, 1st stage, with apicolysis
212 ,, 1st stage, without apicolysis
213 ,, later stages
214 Extrapleural pneumothorax
215 Subphrenic abscess, first stage
216 ,, ,, later stages
217 Mediastinotomy
219 Miscellaneous, extrapleural operations

Intrapleural operations
220, Lobectomy, first stage
221 ,, later stages
222 ,, in one stage
223 Pneumonectomy, first stage
224 ,, later stages
225 ,, in one stage
226 Gunshot wounds, stab wounds, etc.
227 Thoracotomy, thoracentesis
228 Opns. on oesophagus, diaphragm by Th. route
229 Intrapleural operations, miscellaneous

Heart and great vessels
230 Operations on pericardium
231 Operations on heart
232 Operations on great vessels
239 Others, heart and vessels
249 Others, thoracic, unclassifiable

Month		Age		Complications	Sex		Physical Status	
January	1	0—1	0	(Those marked with an asterisk may be used as causes of death)	Male	1	Good	1
February	2	1—9	1		Female	2	Fair	2
March	3	10—19	2				Poor	3
April	4	20—29	3				Serious	4
May	5	30—39	4				Emerg. good	5
June	6	40—49	5				Emerg. poor	6
July	7	50—59	6				Special (Moribund)	7
August	8	60—69	7					
September	9	70—79	8					
October	0	above	9					
November	x	Unknown	x					
December	y							

000 None
001 Inadequate pre-operative information
002 Inadequate post-operative information

Circulatory System

Vascular disease
100 Aortic aneurysm
101 Aneurysm misc.
102 Luetic aortitis
103 Arteriosel., general
104 ,, gangrene
105 Diabetic gangrene.
106 Emboli., misc.
107 Endarteritis oblit.
108 Phlebitis
109 Other vasc. disease
110 Pulm. embolus.*

Functional capacity
120 Functional capacity, I
121 ,, ,, II A
122 ,, ,, II B
123 ,, ,, III
124 Circ. failure*

Heart disease
130 Congenital*
131 Thyrotoxic*
132 Rheumatic*
133 Arteriosclerotic*
134 Luetic*
135 Myocard. degeneration*
136 Coronary disease (pre-op.)
137 Cor. emb. or thromb.*
138 Angina
139 Other

Arrhythmia
140 Heart block*
141 Fibrillation*
149 Other arrhythmia

Shock
150 Anæsthetic*
151 Surgical*

Disorders of rate
160 Tachycardia
161 Bradycardia

Hæmorrhage
170 Hæmorrhage, moderate
171 ,, severe*
172 ,, aneurysms*

Pericardium
180 Pericarditis*
181 Pericardial sac, rupt.*

Miscellaneous
190 Accid. div. important bld. ves.
191 B.P. fall marked, not shock
192 ,, rise, marked
193 Hypertension
194 Hypotension
195 Ascites
196 Oedema
199 Others, miscellaneous

C.N.S.
Mental
200 Apprehension, marked
201 Excitement, marked
202 Irrational
203 Neurosis
204 Psychosis
205 Idiocy
206 Mental degeneration O_2 lack
207 Epilepsy
209 Others

Infections
210 Encephalitis*
211 Meningitis*
212 Epidural abscess*
213 Tetanus*
219 Other infections

Vascular
220 C.N.S. arteriosclerosis*
221 ,, lues*
222 Cereb. emb. or thromb.*
223 Cereb. accident previous
224 Mycotic aneurysm*
225 Sinus thromb.*
229 Other vascular disease, C.N.S.

Paralysis
230 Hemiplegia
231 Myelitis, transverse
232 Section of cord
233 Tumour of cord
234 Meningocoele
235 Paralysis, flaccid
236 ,, spastic
237 Poliomyelitis, ac. ant.
239 Other paralysis

Intracranial pressure
240 Increased intracranial press.
241 Cerebral oedema
242 Concussion*
243 Fractured skull*
244 Hernia cerebri
245 Hernia of medulla*
246 Hydrocephalus*
247 Meningeal hæm.*
250 Cerebral tumour*
251 Cerebral abscess*

Muscular phenomena
260 Convulsions
261 Tremor during anæsthesia
262 Muscle twitchings
263 Other muscular phenomena

Peripheral nerves
270 Paræsthesia
271 ,, foll. nerve block
272 Neuritis, vitamin deficiency
273 ,, toxic, drug, etc.
274 ,, foll. nerve block
275 ,, positional
279 Others

Miscellaneous
290 Headache
291 ,, after L-P
292 Backache after L-P
293 Migraine
294 Pain
295 Autonomic N.S. imbalance
299 Others

Punching

When the card has been coded it is punched with a special hand punch, either directly from the card or from the anæsthetic record chart. If an error is made the card is destroyed and a new one made out. The private code columns are punched first, then starting at the top left-hand corner the rest of the columns are punched in turn. The arrangement of the numbers in the boxes is such that they will clear the machine in time to be punched in their proper columns.

On examination of the completed punched card (Fig. 3) it will be seen that the direction of the punch bears no relation to the figures written on the card, this may appear confusing at first sight until it is realised that no matter where the figures are written on the card they are punched straight across from left to right. For example, the private code columns 1 to 9 are written down on the right-hand side of the card, but are punched in the first nine columns on the left-hand side. The discrepancy between where the figures are written and where they are punched is due to the shape of the punch, which would make the figures invisible to the worker if they were written where they are punched.

Sorting

The cards are sorted by means of a sorting and counting machine provided with twelve pockets corresponding to the holes punched in the cards, and a coffer into which the cards are fed. The machine will sort and count automatically at the rate of 250 cards per minute. But as only one column at a time can be sorted, it is often necessary to perform several sortings in order to obtain the data required. By this procedure it is possible to correlate the facts recorded on the card, and determine the relationship between age, sex, physical state, pre- and post-operative complications operation, anæsthetic agent, surgeon, etc.

The elimination of errors is important, and some form of check is necessary, especially with inexperienced workers, so the following routine work is carried out.

Before the cards are punched they are examined by hand for common errors by two experienced workers. The cards

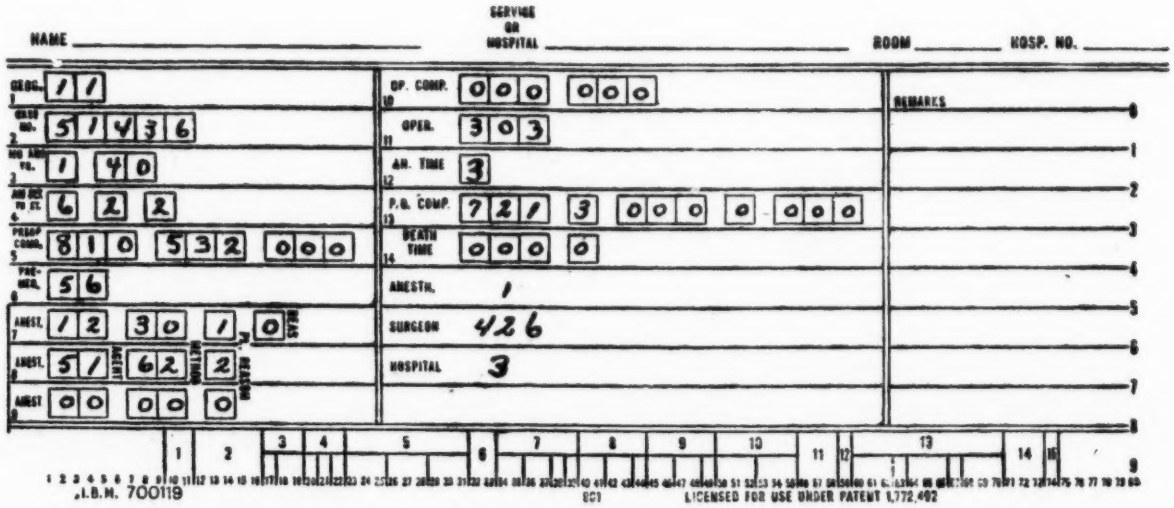

NAME
SERVICE OR HOSPITAL
ROOM
HOSP. NO.
GEOG. 11
AGE 51
SEX 4
RACE 3
AD. DIAG. CODE 622
PREOP COND. 810 532 000
PRE-MED. 56
ANEST. 12 30 1 0
ANEST. 51 62 2
ANEST 00 00 0
OP. COMP. 000 000
OPER. 303
AN. TIME 3
P.S. COMP. 721 3 000 0 000
DEATH TIME 000 0
ANESTH. 1
SURGEON 426
HOSPITAL 3
REMARKS
I.B.M. 700119
LICENSED FOR USE UNDER PATENT 1,772,402

FIG. 3

are run through the sorter set for column 80—any punch hole occurring here indicates an error somewhere on the card. By using the sorter the cards are put in numerical order by their anæsthetic serial number. They are then put in the tabulator, a machine that will print off any required groups of figures on a card. The list is examined by hand to ascertain that it is complete, and that the numbers are consecutive. The cards referring to patients who have died are examined by hand to make sure that the cause of death and post-mortem findings have been accurately coded and punched.

Expense

The equipment is made by the International Business Machine Company, from whom the machines may be hired —not bought. The monthly rental of the hand-punch is about £1; that of the sorter about £10; and the cost of the cards 10s. per 1000.

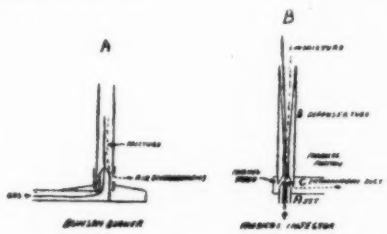
This article has been compiled from notes received from Dr. N. Gillespie, of Wisconsin General Hospital, Madison, U.S.A., to whom I am also indebted for cards and charts reproduced here.

APPLICATION OF THE INJECTOR TO MEDICAL GAS THERAPY*

BY

H. A. E. TALLEY, L.I.B.S.T.

ABOUT a year ago Dr. John Elam approached me on the question of producing an improved nitrous oxide/air apparatus for analgesia in midwifery. The machines produced for this purpose so far invariably suffered from the inherent disadvantage of delivering a mixture of gas and air which varied with the depth of breathing and it was apparent that a method was required which assured a more constant mixture under all conditions. I decided to experiment with the injector principle for this purpose, as I knew that it was already in use in the oxygen apparatus supplied to the German Air Force.



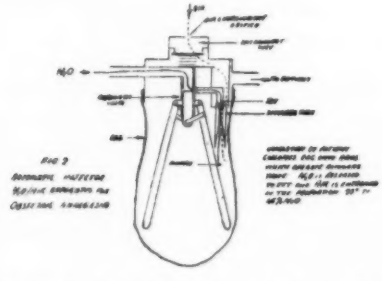
The injector principle is already well known. It is part of a Bunsen burner for instance (Fig. 1A), or the suction apparatus which works by steam, or from a water tap.

Adapted for the administration of medical gases an injector consists of three main parts, the jet "A," diffuser tube "B," and an entrainment duct "C." The gas flow through

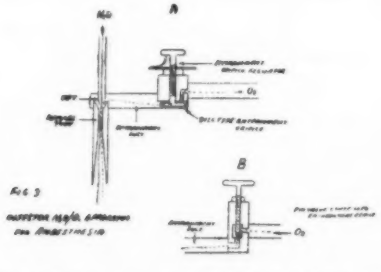
* Read before the Section of Anæsthetists, Royal Society of Medicine, April, 1942.

the main jet is supplied at a higher pressure than in the Bunsen burner owing to the possibility of back pressure, which is frequently developed in anæsthetic machines, interfering with the constant entrainment of a second gas. See Fig. 1B.

A nitrous oxide/air apparatus was produced and this was tested by the Nuffield Department of Anæsthetics at Oxford. In this machine tests showed that the mixture was delivered constantly and accurately and Fig. 2 indicates the general construction of the apparatus.



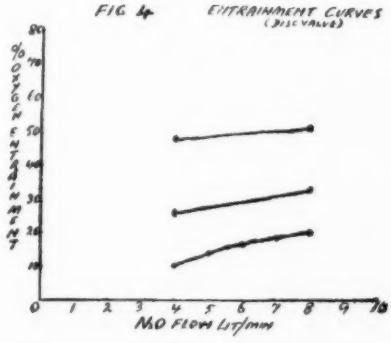
The results obtained with the gas/air machine led me to believe that the injector could be applied for other medical purposes such as the mixing of various gases for anæsthesia or oxygen therapy. A machine was then constructed for



nitrous oxide and oxygen in which a disc type of entrainment regulation orifice was used. Fig. 3A. Tests showed that for a given position of the entrainment regulator the oxygen concentration was not independent of the flow rate

of nitrous oxide but increased with it. Fig. 4 gives the flow of nitrous oxide and oxygen for three positions of the disc type regulator. The lowest curve shows that whilst the oxygen concentration was 10 per cent at a flow of four litres per minute of nitrous oxide, increasing the nitrous oxide to eight litres per minute resulted in the oxygen concentration increasing to 20 per cent.

On the ground of these results Dr. H. G. Epstein of the Nuffield Department of Anæsthetics pointed out to me the necessity of matching the injector with the entrainment regulator. The negative pressure in a given injector for various flow rates of nitrous oxide must be capable of en-



training through the regulator just such volumes of oxygen which will keep the concentration of the gas mixture constant. The disc type of entrainment regulator was unsatisfactory, because the increase of negative pressure developed when doubling the nitrous oxide flow is able to entrain more than double the previous amount of oxygen. From experiments with pin-hole orifices he found that a small needle type of entrainment regulator, Fig. 3B, would give a practical and satisfactory solution.

It was found of importance also that the combination of the jet and diffuser tube could be modified to match the characteristics of the entrainment regulator orifice very closely, and Fig. 5 shows actual results obtained on recent tests with two different jet and diffuser tube combinations in

conjunction with the pin valve entrainment regulator. The accuracy of the figures obtained is dependent on the errors of the rotameters employed in the determinations, but are close enough for all practical purposes.

FIGURE 5

Jet and diffuser tube combination	Nitrous oxide flow litres per minute	Volume percentages oxygen entrainment through small pin valve regulator						Maximum entrainment possible
No. 90 shaped jet	4	2.4	4.8	7.0	11.1	20.0	42.8	65%
and diffuser tube	6		5.5		11.6	20.0	41.2	
with short parallel	8	2.9	5.7	7.7	12.0	21.6		
No. 35 shaped jet	4	2.4	4.8	9.1		20.0	33.3	40%
and diffuser tube	6		5.4	9.4		20.1	34.0	
with long parallel	8	2.7	5.4	9.3		20.0	31.7	

Figures showing proportional mixing of nitrous oxide and oxygen with two different combinations of jet and diffuser tube and pin valve entrainment regulator combinations.

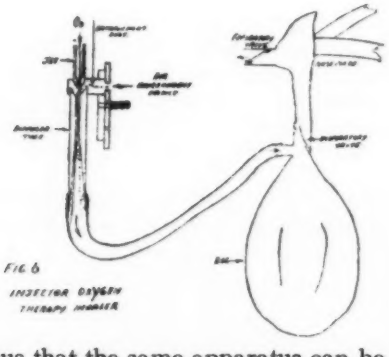


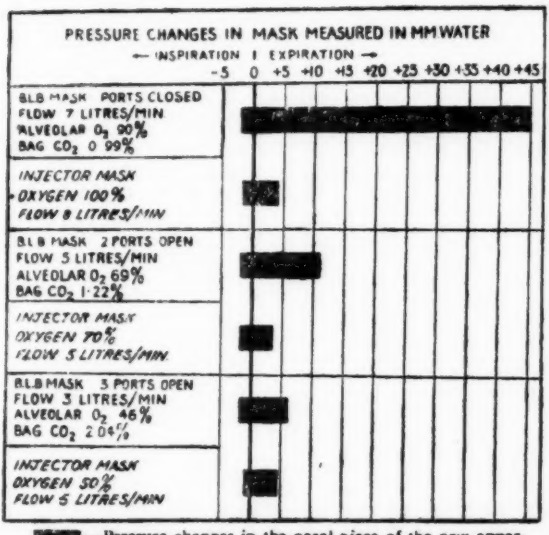
FIG 6

It is obvious that the same apparatus can be used for the combination of any two gases and the absence of ground stopcocks, also the simplicity and robust design, should reduce wear and tear and maintenance to a minimum.

Oxygen Therapy Apparatus.

This apparatus, Fig. 6, was described by Drs. Mitchell and Cowan of the Nuffield Department of Anæsthetics

recently in the *British Medical Journal*. A similar apparatus was described by Drs. Barach and Eckman in the July, 1941, issue of *Anæsthesiology*, and it may be as well to state that my suggestion for this application of the injector was put forward prior to the appearance of the American publication, but pressure of work on the other injector applications delayed matters.



—Pressure changes in the nasal piece of the new apparatus compared with those in the nasal piece of the B.L.B. mask (Summary of Dr. Epstein's figures.)

In this apparatus oxygen is used to entrain air and the entrainment duct is controlled by two ground plates, the outside plate being supplied with a series of holes suitably proportioned to provide oxygen percentages from 40 to 100. As in the tests with the nitrous oxide and oxygen apparatus the hole type of orifice in the entrainment duct guarantees a constant concentration independent of the rate of flow. The inside plate is provided with a large aperture over which any of the holes in the outer plate can be placed by rotation, The holes in the outer plate are marked in the different oxygen

percentages obtained, the figures including the 20 per cent oxygen constituent of the entrained air. The mixture is delivered to the patient by means of a nasal inhaler which consists of a nosepiece and expiratory valve, a large reservoir bag and inspiratory valve, and a supply tube which connects the inhaler to the injector. With this apparatus a constant delivery of any mixture of oxygen and air, or pure oxygen is assured and is delivered into an inhaler designed to reduce resistance to a minimum. Fig. 7 is a reproduction from Drs. Mitchell and Cowan's article of Dr. H. G.

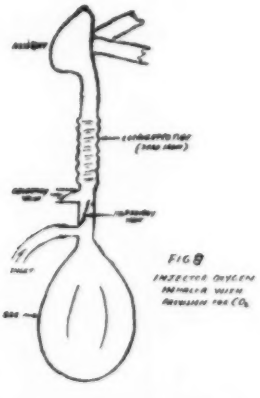


FIG 8

Epstein's figures of resistance in the injector mask as compared with the B.L.B. Inhaler. As no rebreathing is permitted carbon dioxide is efficiently eliminated, but should carbon dioxide be required a rearrangement of the inhaler can be effected by separating the bag and nosepiece by means of a length of corrugated tubing, and transferring the expiratory valve to the position illustrated. Fig. 8.

The amount of carbon dioxide retained depends on the length of corrugated tubing.

Several further applications of the injector are still being investigated, and include a device for mixing oxygen and cyclopropane, also an automatically controlled device for oxygen apparatus for aviation.

I am exceedingly grateful for the opportunity of being

permitted to describe what has been done so far in applying the injector to medical purposes, and I am convinced that the idea has far-reaching possibilities in the mixing and administration of gases.

My thanks and grateful acknowledgment are due to the staff of the Nuffield Department of Anæsthetics, particularly to Dr. H. G. Epstein who has devoted considerable time to the subject, also to Dr. John Elam for stimulating my ten-year-old interest in the matter of accurate apparatus, and to my friends in the profession for their invaluable encouragement.

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Barach, A. L., and Morris Eckman. *Anesthesiology*, 1941, ii, 421.

SERVICE ANÆSTHETISTS

AT the request of some readers, and by courtesy of the authorities, we are publishing a list of anæsthetists who are serving in that capacity with the Army, Navy and Air Force. The number of names being very large we give in this issue a first instalment only of anæsthetists in the R.A.M.C. and in the Royal Navy. The rank mentioned may not, in all instances, be still the correct one.

Adams, Captain E. F.
Alsop, Major A. F.
Ashby, Captain P. T.
Ashwood, Major H. K.

Bain, Major J. W. L.
Bamford, Major J. B.
Barr, Major J. T.
Barrington, Lieut. C. R. G.
Beavor, Major R. A.
Belfrage, Major D. H.
Bereen, Captain J. F.
Binning, Major R. A.
Blackburn, Major J. R.
Blatchley, Major D.
Bodington, Captain J. R.
Boone, Captain W. B.
Boyle, Major A. K.
Braddon, Major I. G.
Brennan, Major H. J.
Broadbent, Major G. S.
Brown, Major A.
Brown, Major A. M.
Buckley, Major J. E.
Burrows, Major J. D.

Carnegie Brown, Captain G.
Chadwick, Major T. H.
Clarke, Major L. T.
Clausen, Major R. J.
Cooper, Captain G. C.
Cope, Major R. W.
Cowrachan, Captain W. E.
Crawford, Major J. H.
Crowley, Major T. F.
Curtiss, Captain E. F.

Daly, Lieut.-Colonel A. S.
Darin, Captain A.
Davison, Major M. H. A.
De Frietas, Major A. J. S.
Devise, Major D.
Dinnis, Major J. W. A.
Dodd, Major H. G.
Dodwell, Major H. B.
Dunn, Captain D. M.
Dunn, Captain J. R.
Dunnill, Captain D. E.

Eason, Major G. A.
Edwards, Major G.
Edwards, Major M.
Edwards, Captain (Miss) P. M.
Ennis, Major G. E.
Emerson, Lieut. J.
Evans, Captain C. D.

Faussett, Captain E. L.
Featherstone, Major H. W.
Finnie, Lieut. W. G.
Fitt, Major T. T.
Fowler, Major E.
Fox, Major G. P.
Friedland, Captain C. M.
Fraulo, Major J.
Fryer, Major T. D. W.

Gabb, Major W. H.
Goldman, Major V. A.
Glazer, Captain P.

Hacking, Major E. B.
Hamerton, Major J. R.

Hamilton, Major C. A. S.
Hammond, Major R. A. E.
Harber, Major M. J.
Harbord, Lieut. R. P.
Harries, Major I. E.
Harris, Major G. A. S.
Harris, Major R. L. H.
Harris, Major T. A. B.
Hartley, Major J. A.
Hayes, Lieut. C.
Headley, Major S. R. T.
Herdman, Major K. N. A.
Hobbes, Major T. H.
Holden, Lieut. C. E.
Howell, Major J.
Howell, Major J. N. H.
Human, Major J. U.

Johnson, Major B. R. M.
Johnson, Major E. F.
Jones, Captain W. M.
Jones, Major I. D.

ROYAL NAVY

Harries, Surg. Lieut.-Com. C. V.
Lees, Surg. Lieut.-Com. J.
Meynell, Surg. Lieut. J. F.
Miles, Surg. Lieut.-Com. (Emerg.) T. F.
Moorby, Surg. Com. A. L.
Ross, Surg. Lieut.-Com. M. G.
Sinclair, Surg. Com. A. D.
Dean, Surg. Lieut.-Com. D. M.
Dinsdale, Tem. Surg. Lieut. T.
Enoch, Act. Surg. Com. R. H.
Johnston, Tem. Surg. Lieut. J. S.
Laidlow, Tem. Surg. Lieut. E. V.
Levy, Surg. Lieut.-Com. S. B.
Locke, Tem. Surg. Lieut. G. B.
Morrice, Tem. Surg. Lieut. G.
Patterson, Surg. Lieut. J.
Ryan, Act. Tem. Surg. Lieut.-Com. J. F.
White, Act. Tem. Surg. Lieut.-Com. A. F.
Woolmer, Act. Surg. Com. R. F.

R.N.V.R.

Blackford, Tem. Surg. Lieut. C. J. G.
Davis, Surg. Lieut. H. de L. N.

ABSTRACTS

"Indications and contra-indications in spinal anæsthesia." L. E. HOLLIS in *Canadian Medical Assoc. Journal.* April 1940, p. 351.

THIS is a very well considered contribution to its subject. The author prefers spinal for all abdominal operations, but he does not use it in the presence of advanced cardio-vascular disease, disease of the central nervous system, syphilis or septicæmia.

He considers subarachnoid block especially indicated for patients with respiratory complications. Age is no barrier to the technique, but "the very young and the very old require most careful consideration before exposing them to the risks of a subarachnoid block." For big thoracic surgery the author believes general anæsthesia to be preferable in most cases, partly because "it abolishes the cough reflex which may spread infection and which in the open chest is inefficient to expel obstructive material from the airway." "Organic changes in renal function are little affected by spinal anæsthesia, hence it is the anæsthetic of choice in kidney diseases." The author finds the results of spinal anæsthesia for Cæsarean section discouraging, and prefers cyclopropane for this operation. The deaths from paralytic ileus and the high incidence of gastro-intestinal complications led him to abandon spinal anæsthesia for Cæsarean section.

"Pentothal sodium anæsthesia." A. G. STANLEY in *New Zealand Medical Journal,* December 1941, p. 359.

The author uses a five per cent solution. Almost the only contraindication to the use of pentothal which he admits is upper respiratory obstruction. For tubercular patients and those suffering from bronchitis or asthma he finds pentothal ideal, contrary to the experience of several other authorities. Pentothal is injected at the rate of one c.c. every

10 to 15 seconds till the patient is asleep or until three c.c. have been given. Then a pause is made for 10 seconds and then one c.c. is injected. This process is repeated till the patient is asleep. A nasal catheter is passed and strapped to the face so that oxygen can be administered throughout the operation. This the author regards as essential for the safety of continuous pentothal anæsthesia. He always uses premedication, morphine and atropine half an hour before operation in short cases; for long operations nembutal gr. 1/2, or morphine 1/6, on the night before, and morphine gr. 1/4, and atropine 1/50 half an hour before operation. The amounts are varied, of course, for the young and the aged. The amounts of pentothal needed for induction varied from 1/4 c.c. to 4+ c.c.; generally the older the patient the less pentothal needed. The greatest amount the author has used was 86 c.c. for a three-hour operation. The patient was conscious in two hours, and had no after effects. The author's conclusions are derived from 515 cases.

"Pre-operative sedation in children." K. BORTHWICK-LESLIE in *Journal Canadian Medical Association.* April 1942, p. 345.

The author rightly draws attention to the deplorable condition of a child brought to the hospital for tonsil and adenoid operation with no previous instruction or sedative. He recommends nembutal or delivinal to be given at home on the evening before operation in the morning. The doses of nembutal prescribed for children are: 2 to $3\frac{1}{2}$ years, gr. $\frac{1}{2}$ to $\frac{3}{4}$; $3\frac{1}{2}$ to 5, gr. $\frac{3}{4}$ to $1\frac{1}{4}$; 5 to 7, gr. 1 to $1\frac{1}{2}$; 7 to 11, gr. $1\frac{1}{2}$ to 2. The delivinal doses are slightly higher. Sodium luminol is used hypodermically in doses ranging from gr. $\frac{1}{8}$ to $\frac{1}{4}$ for children of 2 to 4 years, up to 1 to $1\frac{1}{2}$ grs. for children of ten.

"Post-anæsthetic complications in a military hospital." S. M. CAMPBELL, and R. E. GORDON, in *Canadian Medical Association Journal,* April 1942, p. 347.

This is a useful statistical study based on 2094 anæsthetic records. Reliable conclusions can be drawn when the

patients are all within well defined age limits and are in good health except for their surgical disability. Moreover most of the operations were not of a kind usually associated with high mortality or morbidity rates. Consequently an excellent opportunity was afforded for the study and comparison of anæsthetic agents and techniques.

The figures bear out what is the common experience in military hospitals here, that post-operative chest complications are frequent in these subjects. Their recurrence is shown to be heightened by pre-operative respiratory affections. The authors separate these complications into (1) atelectasis, (2) cough and sputum, and (3) pneumonia. Patients with atelectasis show collapse of a portion of the lung, absent breath sounds or bronchial breathing, respiratory distress and shift of the mediastinum. They have moderate fever of 100° to 103°F. The distress and fever disappear rapidly when treatment is directed to draining away secretions and re-expanding the affected portion of the lung, although the mediastinal shift and the cough and sputum may persist for some days. The patients with cough and sputum, usually labelled "bronchitis," are probably the subjects of patchy atelectasis. They clear up quickly with posturing, coughing and hyperventilation. The increased incidence of post-operative pulmonary complications following operations on the upper abdomen is demonstrated by the authors' figures.

Comparison of patients who had endotracheal intubation with those who were not intubated shows a smaller percentage of post-anæsthetic complications in the intubated group than in the others. Comparison of post-anæsthetic complications following use of spinal agents and cyclopropane for repair of inguinal hernia shows that where no history of previous infection exists the complication after cyclopropane is much less than after spinal analgesia. The incidence of post-anæsthetic pulmonary complications is slightly greater in those who had supplemental anæsthetic with spinal, than with those who had none.

" Pre-operative and post-operative infections of the respiratory tract in relation to inhalation and spinal anæsthesia," in *Archives of Surgery,* January 1942, Vol. 44, No. 1, pp. 41-43.

Abdominal operations were performed in 120 cases, under three types of anæsthesia, i.e. ether, cyclopropane, and spinal; in each case the patient was shown to have some pre-operative chronic respiratory infection. Acute post-operative infections developed in 13.5 per cent of patients after ether anæsthesia, 17.5 per cent after cyclopropane, and 39.5 per cent after spinal anæsthesia.

In a previous study it was found that patients having no pre-operative respiratory infection developed acute post-respiratory infection in 5.8 per cent of cases after ether anæsthesia, 4.9 per cent of cases after cyclopropane, and 7.5 per cent after spinal anæsthesia. It was also shown previously that post-operative respiratory infections were unaffected by age or sex, length of operation, pre-operative complications other than respiratory, the kind or amount of spinal anæsthetic used, premedication or blood-pressure changes.

" The present status of cyclopropane." RALPH M. TOVELL, M.D., and CURTIS B. HICKCOX, M.D., in *Canadian Medical Association Journal,* January 1942, p. 41.

In this article the authors have described most of the recent work done on cyclopropane anæsthesia. Much attention has been paid to the question of cardiac arrhythmias and the effect of premedication on them.

Many anæsthetists advocate the use of a barbiturate as a premedicant. Guedeal advocates the use of an intravenous barbiturate to produce apnœa after which an endotracheal tube is passed and anæsthesia carried on with cyclopropane, respiration being carried on passively. One disadvantage of this method is that emergence delirium often occurs.

Complications during and following cyclopropane anæsthesia are minimal, and their incidence is not increased over that observed with other inhalation agents.

THE DIPLOMA IN ANÆSTHETICS

The following candidates were successful at the last (May) examination for the D.A.:

Helen M. B. Alcock, R. E. Angel, T. H. Baillie, E. J. W. Barnard, J. R. J. Beddard, Stella G. Braithwaite, T. R. M. Bristow, Albert Coleman, Florence K. Costello, J. P. Cuddigan, J. H. Dixon, J. D. Ebsworth, Phyllis M. Edwards, A. J. Evans, Mita Frost, Edith Gilchrist, G. P. Goodwin, J. R. Hindmarsh, A. R. Hunter, Jean E. W. Inglis, Edith S. M. Merry, R. E. J. Pembrey, S. F. Polley, Norah K. Priestley, H. J. Richards, G. K. T. Roche, G. F. Smith, A. E. Ward, Frederick Widlake, and Gertrude A. Willis.

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REVIEW

"Blind Intubation and the Signs of Anæsthesia." (Second edition.) J. U. HUMAN. pp. 135, illustrated. Price 5*s*. John Bale Medical Publications Ltd.

IN the second edition of this book, section one deals with the signs of anæsthesia. Besides describing the variations in size of the pupil, the eyelid reflex, the roving eyeball, the corneal reflex, swallowing and vomiting, the author describes the eyelash reflex and chin retraction. The former is elicited by gently brushing the tips of the eyelashes outwards with the ball of a finger, and consists of a slight contraction of the orbicularis palpebrarum. The latter consists of a downward movement of trachea and larynx with each inspiration, the chin being pulled downwards at the same time. The alterations in respiration are described as being an unreliable indication of the depth of anæsthesia.

Section two deals with intubation. The advantages of using an endotracheal tube as an airway in abdominal cases are described. A central pointed endotracheal tube is recommended in preference to the Magill's tube, as the author considers there is a greater danger of trauma occuring with the latter; this is contrary to our experience. For beginners the author recommends the semi-direct method of intubation—and considers that if the tip of the epiglottis is lifted, and the tube manipulated into the space behind the epiglottis, it is not necessary for the anæsthetist to see the vocal cords. For blind intubation he recommends that CO_2 should be added to the anæsthetic mixture, and when the patient is still "light," with all muscles tense, the tube can be inserted blindly with the utmost ease.

The third section deals with premedication, all the drugs used for this purpose are briefly mentioned.

The author, in dealing with opiates and scopolamine, recommends that omnipon 2/3 with scopolamine 1/150

should be given in preference to the usual dose of omnipon gr. 1/3 and scopolamine gr. 1/150. He also states that these drugs should not be given to children but gives no reason for this theory. Children who have high B.W.R.'s tolerate these drugs extremely well—and in a series of over 2000 cases we have found fractional doses of these drugs a most satisfactory form of premedication and more reliable than the barbiturate derivatives which, as the author points out, so often cause post-operative restlessness.

There is a short chapter on gas cylinders, and another excellent chapter on induction of open ether anæsthesia.

We feel sure that this small book, which contains many useful hints and sound advice, cannot fail to benefit the anæsthetic student for whom it was chiefly written.

E.C.